PB81-117269

# Ambient Water Quality Criteria for Acenaphthene

(U.S.) Environmental Protection Agency
Washington, DC

Oct 80

U.S. Department of Commerce
National Technical Information Service
NTIS.

United States
Environmental Protection
Agency

Office of Water
Regulations and Standards
Criteria and Standards Division
Washington DC 20460

EPA 440/5-80-015
October 1980

EPA

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PB81-117269

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NATIONAL TECHNICAL
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U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

**TECHNICAL REPORT DATA**
*(Please read Instructions on the reverse before completing)*

| 1. REPORT NO. | 2. | 3. RECIPIENT'S ACCESSION NO. PB81 117269 |
|---|---|---|
| 4. TITLE AND SUBTITLE<br>Ambient Water Quality Criteria for:<br>Acenaphthene | | 5. REPORT DATE<br>October, 1980<br>6. PERFORMING ORGANIZATION CODE<br>NTISEPAOWP |
| 7. AUTHOR(S)<br>Environmental Criteria and Assessment Office | | 8. PERFORMING ORGANIZATION REPORT NO.<br>EPA-440/5-80-015 |
| 9. PERFORMING ORGANIZATION NAME AND ADDRESS<br>Office of Water Regulations and Standards<br>Criteria and Standards Division<br>U.S. Environmental Protection Agency<br>Washington, D.C. 20460 | | 10. PROGRAM ELEMENT NO.<br>11. CONTRACT/GRANT NO. |
| 12. SPONSORING AGENCY NAME AND ADDRESS | | 13. TYPE OF REPORT AND PERIOD COVERED<br>14. SPONSORING AGENCY CODE |

15. SUPPLEMENTARY NOTES
Supersedes draft document PB 296 782

16. ABSTRACT
Section 304(A) of the Clean Water Act [33 U.S.C. 1314(A)], requires EPA to publish and periodically update water quality criteria. These criteria are to reflect the latest scientific knowledge on the identifiable effects of pollutants on public health and welfare, aquatic life, and recreation. This report presents water quality criteria for Acenaphthene. It presents concentration criteria for the protection of freshwater and saltwater aquatic life. It presents 'safe' concentrations for humans, and in the case of suspect or proven carcinogens, gives various levels of incremental cancer risk. A Section 304(A) water quality criterion is a qualitative or quantitative estimate of the concentration of a water constituent or pollutant in ambient waters which, when not exceeded, will ensure a water quality sufficient to protect a specified water use. Under the Act a criterion is a scientific entity, based solely on data and scientific judgment. It does not reflect considerations of economic or technological feasibility nor is it a water quality standard and in itself has no regulatory effect.

17. KEY WORDS AND DOCUMENT ANALYSIS

| a. DESCRIPTORS | b. IDENTIFIERS/OPEN ENDED TERMS | c. COSATI Field/Group |
|---|---|---|
| | | |

| 18. DISTRIBUTION STATEMENT | 19. SECURITY CLASS (This Report) | 21. NO. OF PAGES |
|---|---|---|
| For Public Release | Unclassified | |
| | 20. SECURITY CLASS (This page)<br>Unclassified | 22. PRICE |

EPA Form 2220-1 (9-73)

# AMBIENT WATER QUALITY CRITERIA FOR

# ACENAPHTHENE

Prepared By
U.S. ENVIRONMENTAL PROTECTION AGENCY

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# FOREWORD

Section 304 (a)(1) of the Clean Water Act of 1977 (P.L. 95-217), requires the Administrator of the Environmental Protection Agency to publish criteria for water quality accurately reflecting the latest scientific knowledge on the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of pollutants in any body of water, including ground water. Proposed water quality criteria for the 65 toxic pollutants listed under section 307 (a)(1) of the Clean Water Act were developed and a notice of their availability was published for public comment on March 15, 1979 (44 FR 15926), July 25, 1979 (44 FR 43660), and October 1, 1979 (44 FR 56628). This document is a revision of those proposed criteria based upon a consideration of comments received from other Federal Agencies, State agencies, special interest groups, and individual scientists. The criteria contained in this document replace any previously published EPA criteria for the 65 pollutants. This criterion document is also published in satisifaction of paragraph 11 of the Settlement Agreement in Natural Resources Defense Council, et. al. vs. Train, 8 ERC 2120 (D.D.C. 1976), modified, 12 ERC 1833 (D.D.C. 1979).

The term "water quality criteria" is used in two sections of the Clean Water Act, section 304 (a)(1) and section 303 (c)(2). The term has a different program impact in each section. In section 304, the term represents a non-regulatory, scientific assessment of ecological effects. The criteria presented in this publication are such scientific assessments. Such water quality criteria associated with specific stream uses when adopted as State water quality standards under section 303 become enforceable maximum acceptable levels of a pollutant in ambient waters. The water quality criteria adopted in the State water quality standards could have the same numerical limits as the criteria developed under section 304. However, in many situations States may want to adjust water quality criteria developed under section 304 to reflect local environmental conditions and human exposure patterns before incorporation into water quality standards. It is not until their adoption as part of the State water quality standards that the criteria become regulatory.

Guidelines to assist the States in the modification of criteria presented in this document, in the development of water quality standards, and in other water-related programs of this Agency, are being developed by EPA.

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ACKNOWLEDGEMENTS

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CRITERIA DOCUMENT

ACENAPHTHENE

## CRITERIA

### Aquatic Life

The available data for acenaphthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,700 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acenaphthene to sensitive freshwater aquatic animals but toxicity to freshwater algae occur at concentrations as low as 520 µg/l.

The available data for acenaphthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentrations as low as 970 and 710 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 500 µg/l.

### Human Health

Sufficient data are not available for acenaphthene to derive a level which would protect against the potential toxicity of this compound. Using available organoleptic data, for controlling undesirable taste and odor quality of ambient water, the estimated level is 0.02 mg/l. It should be recognized that organoleptic data, as a basis for establishing a water quality criteria, have limitations and have no demonstrated relationship to potential adverse human health effects.

## INTRODUCTION

Acenaphthene (1,2-dehydro-acenaphtnylene or 1,8-ethylene-naphthalene) occurs in coal tar produced during the high temperature carbonization or coking of coal. It is used as a dye intermediate, in the manufacture of some plastics, as an insecticide and fungicide, and has been detected in cigarette smoke and gasoline exhaust condensates. Acenaphthene is a polynuclear aromatic hydrocarbon with a molecular weight of 154 and a formula of $C_{12}H_{10}$.

The compound is a white crystalline solid at room temperature with a melting range of 95 to 97°C and a boiling range of 278 to 280°C (Lidner, 1931). The vapor pressure is less than 0.02 mm Hg. Acenaphtnene is soluble in water (100 mg/l), but solubility is greater in organic solvents such as ethanol, toluene, and chloroform.

Acenaphthene will react with molecular oxygen in the presence of alkali-earth bromides to form acenaphthequinone (Digurov, et al. 1970). In the presence of alkali-earth metal hydroxides, acenaphthene reacts with ozone to produce 1,8-naphthaldehyde carboxylic acid (Menyailo, et al. 1971). Acenaphthalene can be oxidized to aromatic alcohols and ketones using transition metal compounds as catalysts (Yakobi, 1974). Acenaphtnene is stable under laboratory conditions and resists photochemical degradation in soil (Medvedev and Davydow, 1972).

Laboratory experimentation points out the possibility of limited metabolism of acenaphthene to napthalic acid and napatholic anhydride.

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Aquatic Life Toxicology*

## INTRODUCTION

The data base for acenaphthene and freshwater and saltwater organisms is limted to a few acute toxicity tests under static conditions with unmeasured concentrations. A bioconcentration test has been conducted for 28 days and the depuration rate was determined. An embryo-larval test with the sheepshead minnow has been conducted.

## EFFECTS

### Acute Toxicity

An acute test with Daphnia magna resulted in a 48-hour $EC_{50}$ of 41,200 µg/l and, when the bluegill was exposed to acutely lethal concentrations of acenaphthene, the resulting 96-hour $LC_{50}$ value was 1,700 µg/l (U.S. EPA, 1978) (Table 1).

For the mysid shrimp (U.S. EPA, 1978) the 96-hour $LC_{50}$ is 970 µg/l, and the 96-hour $LC_{50}$ value for the sheepshead minnow is 2,230 µg/l (Table 1).

### Chronic Toxicity

The acute-chronic ratio for the sheepshead minnow is small (3.1). The 96-hour $LC_{50}$ was 2,230 µg/l (Table 1) and the geometric mean of the noeffect and effect concentrations was 710 µg/l (Table 2).

No other chronic data are available.

*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life and Its Uses in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are calculations for deriving various measures of toxicity as described in the Guidelines.

Plant Effects

The freshwater alga, Selenastrum capricornutum, appears to be rather sensitive with 96-hour $EC_{50}$ values for chlorophyll a and cell numbers of 530 and 520 ug/l, respectively (Table 3).

The saltwater alga, Skeletonema costatum, is more sensitive than the sheepshead minnow and the mysid shrimp with a 96-hour $EC_{50}$ value for chlorophyll a and cell numbers of 500 ug/l.

Residues

The bluegill accumulated acenaphthene during a 28-day exposure (U.S. EPA, 1978) and the bioconcentration factor was 387 using $^{14}C$-acenaphthene and thin-layer chromatography for verification (Table 4). The half-life of this chemical in the whole body was less than 1 day.

Summary

The bluegill was much more sensitive to acenaphthene than the cladoceran, Daphnia magna; 50 percent effect concentrations are 1,700 and 41,200 ug/l, respectively. The freshwater alga, Selenastrum capricornutum, was more sensitive than the fish species with a 96-hour $EC_{50}$ of 520 ug/l for cell number. The bioconcentration factor for the bluegill and acenaphthene is 387 with a tissue half-life of less than 1 day.

Contrary to the pattern with freshwater species, the invertebrate species, Mysidopsis bahia, was more sensitive (96-hour $LC_{50}$ of 970 ug/l) than the sheepshead minnow (96-hour $LC_{50}$ of 2,230 ug/l). The saltwater alga, Skeletonema costatum, was sensitive to acenaphthene with a 96-hour $EC_{50}$ of 500 ug/l for both chlorophyll a and cell number. The acute-chronic ratio for the sheepshead minnow is quite small (3.1) and indicates that successful growth and reproduction occurs at a concentration close to one that causes mortality.

## CRITERIA

The available data for acenaphthene indicate that acute toxicity to freshwater aquatic life occurs at concentrations as low as 1,700 µg/l and would occur at lower concentrations among species that are more sensitive than those tested. No data are available concerning the chronic toxicity of acenaphthene to sensitive freshwater aquatic animals but toxicity to freshwater algae occur at concentrations as low as 520 µg/l.

The available data for acenaphthene indicate that acute and chronic toxicity to saltwater aquatic life occur at concentraztions as low as 970 and 710 µg/l, respectively, and would occur at lower concentrations among species that are more sensitive than those tested. Toxicity to algae occurs at concentrations as low as 500 µg/l.

Table 1. Acute values for acenaphthene (U.S. EPA, 1978)

| Species | Method[a] | LC50/EC50 (µg/l) | Species Acute Value (µg/l) |
|---|---|---|---|
| | FRESHWATER SPECIES | | |
| Cladoceran, *Daphnia magna* | S, U | 41,200 | 41,200 |
| Bluegill, *Lepomis macrochirus* | S, U | 1,700 | 1,700 |
| | SALTWATER SPECIES | | |
| Mysid shrimp, *Mysidopsis bahia* | S, U | 970 | 970 |
| Sheepshead minnow, *Cyprinodon variegatus* | S, U | 2,230 | 2,230 |

[a] S = static, U = unmeasured

No Final Acute Values are calculable since the minimum data base requirements are not met.

Table 2. Chronic values for acenaphthene (U.S. EPA, 1978)

| Species | Method* | Limits (µg/l) | Chronic Value (µg/l) |
|---|---|---|---|
| SALTWATER SPECIES | | | |
| Sheepshead minnow, Cyprinodon variegatus | E-L | 520-970 | 710 |

* E-L = embryo-larval

Acute-Chronic Ratio

| Species | Chronic Value (µg/l) | Acute Value (µg/l) | Ratio |
|---|---|---|---|
| Sheepshead minnow, Cyprinodon variegatus | 710 | 2,230 | 3.1 |

Table 3. Plant values for acenaphthene (U.S. EPA, 1978)

| Species | Effect | Result (µg/l) |
|---|---|---|
| **FRESHWATER SPECIES** | | |
| Alga, *Selenastrum capricornutum* | Chlorophyll *a* 96-hr EC50 | 530 |
| Alga, *Selenastrum capricornutum* | Cell numbers 96-hr EC50 | 520 |
| **SALTWATER SPECIES** | | |
| Alga, *Skeletonema costatum* | Chlorophyll *a* 96-hr EC50 | 500 |
| Alga, *Skeletonema costatum* | Cell counts 96-hr EC50 | 500 |

Table 4. Residues for acenaphthene (U.S. EPA, 1978)

| Species | Tissue | Bioconcentration Factor | Duration (days) |
|---|---|---|---|
| | FRESHWATER SPECIES | | |
| Bluegill, *Lepomis macrochirus* | whole body | 387 | 28 |

## REFERENCES

U.S. EPA. 1978. In-depth studies on health and environmental impacts of selected water pollutants. Contract No. 68-01-4646.

## Mammalian Toxicology and Human Health Effects

### EXPOSURE

### Ingestion from Water

Acenaphthene has been detected in the effluents from petrochemical, pesticide, and wood preservative industries by U.S. EPA monitoring studies (U.S. EPA, 1978b). A survey of organic chemical monitoring data from a variety of published and unpublished sources indicated that acenaphthene had been identified in 11 studies (U.S. EPA, 1976). Seven of these studies analyzed effluent from petrochemical or wood preserving plants, while two identified the chemical in finished drinking water, and another study found it in a river sample. An analysis of the settling pond water from a wood preserving plant showed acenaphthene present at a level of 0.2 mg/l (U.S. EPA, 1973). Acenaphthene was also identified by two Russian authors as one of several organic compounds found in wastewater as a by-product of coke manufacturing (Andreikova and Kogan, 1977).

In an examination of water extracted by macroreticular resins from a contaminated well in Ames, Iowa, investigators isolated acenaphthene at a level of 1.7 ppm (Burnham, et al. 1972). Identification was verified by comparison with mass spectrum, chromatography retention time, and ultraviolet spectrum of a standard. The authors (Burnham, et al. 1972) noted that the contamination is believed to be the result of residue from a coal gas plant which may have leached into the aquifer after the plant closed in 1930. Meijers and Van der Leer (1976) detected acenaphthene by gas chromatography in a 20 liter sample of water from the river Maas in the Netherlands. Although not quantified by the authors, acenaph-

thene was a minor constituent of the polycyclic aromatic hydrocarbons (PAH) mixture identified in the water. Acenaphthene has a low solubility in water, but its presence in water may be significant due to possible adsorption on particulates.

Ingestion from Food

Only one study (Onuska, et al. 1976) was found on the occurrence of acenaphthene in foods. Levels of $\geq$3.2 µg acenaphthene/kg (the detection limit) were reportedly identified in the tissues of shellfish of an unspecified species and location. Relative to other PAH detected in this sample, the amount of acenaphthene was small.

A bioconcentration factor (BCF) relates the concentration of a chemical in aquatic animals to the concentration in the water in which they live. The steady-state BCF for a lipid-soluble compound in the tissues of various aquatic animals seem to be proportional to the percent lipid in the tissue. Thus, the per capita ingestion of a lipid-soluble chemical can be estimated from the per capita consumption of fish and shellfish, the weighted average percent lipids of consumed fish and shellfish, and a steady-state BCF for the chemical.

Data from a recent survey on fish and shellfish consumption in the United States were analyzed by SRI International (U.S. EPA, 1980). These data were used to estimate that the per capita consumption of freshwater and estuarine fish and shellfish in the United States is 6.5 g/day (Stephan, 1980). In addition, these data were used with data on the fat content of the edible portion of the same species to estimate that the weighted average percent

lipids for consumed freshwater and estuarine fish and shellfish is 3.0 percent.

A measured steady-state BCF of 387 was obtained for acenaphthene using bluegills (U.S. EPA, 1978a). Similar bluegills contained an average of 4.8 percent lipids (Johnson, 1980). An adjustment factor of 3.0/4.8 = 0.625 can be used to adjust the measured BCF from the 4.8 percent lipids of the bluegill to the 3.0 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average BCF for acenaphthene and the edible portions of all freshwater and estuarine aquatic organisms consumed by Americans is calculated to be 387 x 0.625 = 242.

Inhalation

Acenaphthene has been identified as one of many polycyclic aromatic hydrocarbons (PAH) in gasoline exhaust condensate (Grimmer, et al. 1977) and cigarette smoke condensate (Harke, et al. 1976; Severson, et al. 1976). However, no estimates have been made of the degree of exposure to acenaphthene that occurs to individuals inhaling cigarette smoke or gasoline exhaust.

A 420,000 $ft^3$ sample of air in Sydney, Australia, was found to contain 3.9 ppm of solid acenaphthene, or 0.07 µg/100 $m^3$ (Cleary, 1962), indicating that individuals in urban environments may be exposed to measurable levels of acenaphthene.

Dermal

Pertinent data could not be located in the available literature on dermal exposure to acenaphthene.

## PHARMACOKINETICS

### Absorption and Distribution

Pertinent information could not be located in the available literature on the absorption and distribution of acenaphthene.

### Metabolism

Chang and Young (1943) isolated, by several methods, the anhydride of naphthalene-1,8-dicarboxylic acid from the urine of two groups of male white rats administered acenaphthene orally. One group of rats was fed twice a day on a stock diet containing 1 percent acenaphthene; a second group was dosed by gavage on alternate days with 1 ml of a fine suspension of 0.1 g acenaphthene in dilute starch solution. The authors suggested the possibility that the naphthalic anhydride is a decomposition product of conjugated metabolites that arose from the acid used in the extraction procedure, rather than a metabolic product of acenaphthene. Acenaphthene was not detected in the urine of the rats.

Aside from this study, no other data were found concerning the metabolism of acenaphthene.

### Excretion

Acenaphthene was not found in the acidified urine of rats dosed orally with acenaphthene (Chang and Young, 1943). No other data are available on the excretion of acenaphthene.

## EFFECTS

### Acute, Subacute, and Chronic Toxicity

Very little is known about the human toxicity of acenaphthene. It is irritating to skin and mucous membranes, and may cause vomiting if swallowed in large quantities (Sax, 1975).

Similarly, limited data are available on the toxic effects of acenaphthene in mammals. Knobloch, et al. (1969) investigated the acute and subacute toxic effects of acenaphthene in rats and mice. Acenaphthene at 2 g/kg body weight administered orally in olive oil to seven young rats (sex not specified) daily for 32 days caused loss of body weight and changes in peripheral blood, increased aminotransferase levels in blood serum, and produced mild morphological damage to both the liver and kidney. A $LD_{50}$ of 10 g/kg was reported for rats and 2.1 g/kg for mice. The authors (Knobloch, et al. 1969) noted that the morphological damage to the kidney and liver was greater when acenaphthene was administered in a subacute manner than when an acute dose was given. After 32 days of treatment the animals showed mild bronchitis and localized inflammation of the peribronchial tissue.

In another toxicity study, Reshetyuk, et al. (1970) exposed 100 rats to a 5-month chronic inhalation of acenaphthene at a level of $12 \pm 1.5$ mg/m$^3$ for four hours a day, six days per week. Toxic effects on the blood, lungs, and glandular constituents were reported. The bronchial epithelium showed hyperplasia and metaplasia, which may have been symptoms of the pneumonia that killed a large number of animals. However, no signs of malignancy appeared during the 8-month post-exposure observation period. Reshetyuk, et al. (1970) also reported a $LD_{50}$ of $600 \pm 60$ mg/kg for rats given intraperitoneal injections of acenaphthene. It must be pointed out, however, that the lack of reported controls, as well as the inadequate and confusing description of methods, make this study unsuitable as the basis for a criterion.

Gershbein (1975) investigated the effect of acenaphthene and many other hydrocarbons upon the degree of liver regeneration in partially hepatectomized male rats. Acenaphthene in peanut oil was injected subcutaneously into one group of animals daily for seven days following surgery for a total dose of 5 to 20 mmol/kg. A second group of animals was administered the chemical as part of the diet at 0.03 and 0.10 percent (by weight). Ten days following the surgical treatment, all animals were sacrificed and the liver weights determined. Liver regeneration was significantly (p 0.01) accelerated in both the injection-treated animals and the higher oral dose group. A third group of rats was injected with acenaphthene three times and then sacrificed 72 hours after surgery. Among all those exposed in this manner to five polycyclic hydrocarbons, acenaphthene-treated animals were the only animals showing a significant acceleration of liver regeneration. These results are in contrast to an earlier study by Gershbein (1958), in which a low dose of 4.6 mmol/acenaphthene/kg did not result in a significant liver regeneration acceleration. In the 1958 study, only a dose of 31.8 mmol/kg induced a significant regeneration.

Although the toxic effects of acenaphthene are not well documented, the reactions of humans to an odor from an aqueous solution of the chemical, which may result in rejection of the contaminated water, have been investigated. In a study of the odor thresholds of organic pollutants (Lillard and Powers, 1975), a panel of 14 judges detected acenaphthene at a mean threshold of 0.08 ppm, with a range of 0.02 to 0.22 ppm. Using these threshold values, extreme value calculations were performed to predict levels of acenaphthene

that a certain percentage of the population could detect. These calculations are shown as follows:

| Percent of Population Able to Detect Odor | Concentration of Acenaphthene (ppm) |
|---|---|
| 20 | $2.6 \times 10^{-2}$ |
| 10 | $1.4 \times 10^{-2}$ |
| 1 | $1.9 \times 10^{-3}$ |
| 0.1 | $2.1 \times 10^{-4}$ |

Synergism and/or Antagonism

Two studies were conducted to investigate the effect of acenaphthene on the activity of dimethylnitrosamine demethylase (DMN-demethylase), the liver enzyme that demethylates dimethylnitrosamine (DMN), a known carcinogen. Argus, et al. (1971) and Arcos, et al. (1976) injected male weanling rats intraperitoneally with acenaphthene at a concentration equimolar to 40 mg of 20-methylcholanthrene/kg body weight. Twenty-four hours later, the animals were sacrificed and the liver microsomes assayed for DMN-demethylase activity. Acenaphthene showed a 0 percent (Argus, et al. 1971) and a 5 percent (Arcos, et al. 1976) depression of the DMN-demethylase levels over control rats with the same birth date. The difference in enzyme activity for the two studies may have been due to a modification of formaldehyde detection methods (Venkatesan, et al. 1968). Arcos, et al. (1976) noted that demethylation is a requirement for carcinogenesis by DMN and, thus, it is possible that acenaphthene may slightly inhibit DMN carcinogenesis.

Buu-Hoi and Hien-Do-Phouc (1969) investigated the effect of acenaphthene and other PAH on the activity of zoxazolamine hydroxylase. Male Wistar rats were injected intraperitoneally with

20 mg/kg acenaphthene in corn oil, followed one week later by 90 mg/kg zoxazolamine. The mean paralysis time of treated rats was found to be significantly greater ($p < 0.01$) than that of vehicle-injected animals. The authors interpreted these results as an indication that acenaphthene retards the detoxification of zoxazolamine, which ordinarily proceeds via hydroxylation.

Teratogenicity

Pertinent data could not be located in the available literature concerning the teratogenicity of acenaphthene.

Mutagenicity

The only data found on the mutagenicity of acenaphthene were four studies using microorganisms as the indicator system (Clark, 1953a,b; Gibson, et al. 1978; Guerin, et al. 1978). No mutagenicity was observed in any of the procedures used. Clark (1953a) studied the effect of acenaphthene on the recombination rate of two auxotrophic Escherichia coli strains. Acenaphthene was found to have no appreciable effect upon the recombination rate of either strain, as indicated by the low level of prototroph induction. Acenaphthene did induce pleomorphism, but not the filamentous "large" form which has been correlated with gene recombination. No metabolic activation was used in this study and the dose of acenaphthene administered was not specified. In a later study, Clark (1953b) tested acenaphthene for mutagenicity by exposing Micrococcus pyogenes var. aureus strain FDA209 to a saturated solution of acenaphthene in a water-based nutrient broth without a metabolic

activation system. When induction of mutants resistant to penicillin or streptomycin was assessed, acenaphthene did not demonstrate any mutagenic effects.

Two mutagenicity studies performed using Salmonella typhimurium gave negative or inconclusive results. Guerin, et al. (1978) isolated an acenaphthene-containing aromatic subfraction from shale-derived crude oil and tested it for mutagenicity using S. typhimurium TA98. No increases were observed with or without rat liver activation. Gibson, et al. (1978) exposed S. typhimurium strains to 200 to 2,000 µg of acenaphthene dissolved in dimethylsulfoxide after first irradiating the acenaphthene samples with $^{60}Co$ to simulate (or replace) liver microsome activation. Unfortunately, the results were erratic with major toxicity observed at all dose levels tested. This toxicity obscured any assessment of mutagenicity.

The studies discussed above were the only ones found in the literature that examined the mutagenic potential of acenaphthene. A fifth study (Harvey and Halonen, 1968) examined the binding of acenaphthene to a variety of biologically important compounds as part of an unsuccessful attempt to correlate the nucleoside-binding activity of various chemicals with their carcinogenic potential. Acenaphthene showed significant binding constants for caffeine and riboflavin, but not for nucleosides.

Other Cellular Effects

The most thoroughly investigated effect of acenaphthene is its ability to produce nuclear and cytological changes in microbial and plant species. Most of these changes, such as an increase in cell

size and DNA content, are associated with disruption of the spindle mechanism during mitosis and the resulting induction of polyploidy. While there is no known correlation between these effects and the biological impact of acenaphthene on mammalian cells, these effects are reported in this document because they are the only substantially investigated effects of acenaphthene.

Ten experiments examining the effect of acenaphthene on plants and eight others involving the effects upon microorganisms are discussed in the following sections. A summary of these data is presented in Table 1.

Plants: Kostoff (1938a) exposed Nicotiana longiflora shoots to vapor from acenaphthene crystals and examined the shoots for effects on mitosis and/or meiosis. The exposure induced tetraploid and octaploid shoots, which produced seeds of new polyploid plants. The polyploidizing effect of acenaphthene vapor increased with increases in the length of exposure or the number of particles used. Kostoff (1938b) also tested the effect of acenaphthene on the branches of floral buds of nine Nicotiana species. Meiosis in the buds proceeded abnormally also, with the bivalent chromosomes failing to arrange correctly on the equatorial plate. They tended to spread into the cytoplasm singly or in groups, resulting in a variable number of chromosomes per nuclei at the end of the second division. Fifty to one-hundred percent of the pollen produced by the end of meiosis was abortive.

In the same study, Kostoff (1938b) covered germinating seeds from a variety of plants with acenaphthene crystals to study the effects on mitosis. Cereals and grasses (wheat, rye, barley, oat,

TABLE 1

Summary of Polyploid and Other Mitotic Effects Induced by Acenaphthene in Plants and Microorganisms

| Organism | Treatment | Effects Noted | Reference |
|---|---|---|---|
| Plants: | | | |
| *Nicotiana* shoots | Vapor | Stable polyploidy; abnormal, abortive meiosis | Kostoff, 1938a,b |
| Cereal, grass legume, and compositae seeds | Crystals (4-12 days) | Abnormal mitosis, spindle mechanism inhibited | Kostoff, 1938b |
| Cherry-mazzard hybrid seeds | Powder (10 hours) | Seed germination and growth inhibited; no polyploidy | Zhukov, 1971 |
| *Allium cepa* L. | Saturated solution (2-5 days) | Chromosome fragmentation, polyploidy | D'Amato, 1949 |
| *Allium cepa* L., *A. sativum* | Treatment unspecified | Frequency of division retarded, multiple prophase | Mookerjee, 1973 |
| *Allium fistulosum*, *Colchicum* roots | Crystals wrapped in moist filter paper (4-20 days) | C-mitosis, polyploidy, root-tip swellings | Levan, 1940 |
| *Allium* root cells | Vapor (12-96 hours) | Random cell wall development | Mesquita, 1967 |

TABLE 1 (continued)

| Organism | Treatment | Effects Noted | Reference |
|---|---|---|---|
| Binucleate pollen | Vapor | Spindle inhibited, division stopped at metaphase | Dyer, 1966 |
| *Tradescantia* pollen | Vapor | Spindle disturbed | Swanson, 1940 |
| *Tradescantia* stamen hairs | Saturated solution (2-4.5 hours) | No polyploidy, no chromosomes in metaphase | Nebel, 1938 |
| Fungi: | | | |
| Basidomycetes | Vapor | Mitotic frequency decrease; growth, pigment formation, differentiation and morphology changes | Hoover, 1972 |
| *Basidiobolus ranarum* hyphae | Vapor (6-18 hours) | Alterations in nuclear division | Hoover and Liberta, 1974 |
| *Pythium aphanidermatum* hyphae | Vapor or supersaturated solution (12 hours) | Nuclear division arrested; pyknosis | Seshadri and Payak, 1970 |
| Yeast | $10^{-1}$ to $3 \times 10^{-7}$ mol solution | No lethality or c-mitosis | Levan and Sandwall, 1943 |
| *Candida scottii* | 0.2-1.0% agar | Increase in cell size, nucleus, DNA content | Imshenetsky, et al. 1966 |

TABLE 1 (continued)

| Organism | Treatment | Effects Noted | Reference |
|---|---|---|---|
| Bacteria: | | | |
| *Mycobacterium rubrum* | Vapor from 10-20 mg crystals | Elongation and thickening of cells; unstable polyploidy | Imshenetsky and Zhil'tsova, 1973 |
| *Rhizobium* | Vapor | Increase in DNA content; change in biochemical properties | Avvakumova, et al. 1975 |
| Algae: | | | |
| *Chara globularis*; *Nitella flagelliformis* | Saturated solution (12-120 hours) | Number of cells in mitosis reduced; chromosomes clumped at metaphase; chromosomes doubled | Sarma and Tripathi, 1976a,b |

maize, and rice) showed slow growth and abnormal roots and leaf formations after 4 to 8 days. Legumes evidenced these effects after 6 to 12 days, while Compositae reacted in a time period midway between the other two groups. Mitosis in these seedlings proceeded abnormally; the spindle mechanism was inhibited and the chromosomes were not arranged on the equatorial plate. Failure of the chromosomes to move to the poles resulted in polyploidy.

Zhukov (1971) investigated the effect of acenaphthene on plant seeds. He treated "cherry-mazzard hybrid" seeds with acenaphthene powder for 10 hours. Seed germination and seedling growth were inhibited, but no polyploidal cells were found in the plant roots.

Four investigators performed experiments with acenaphthene and Allium plants. When treated with saturated solutions of acenaphthene in either tap or distilled water for 2 to 5 days, Allium cepa demonstrated intense chromosome fragmentation (D'Amato, 1949). Fragmenting effects on diploid and polyploidized nuclei in the resting stage were noted, as were centromere effects on the metaphase chromosomes and, occasionally, on chromatids at anaphase. In a later study (Mookerjee, 1973), acenaphthene exposure (concentration unspecified) was found to retard the frequency of division of Allium cepa and Allium sativum. Multiple prophase was observed in A. cepa.

Levan (1940) dusted Allium fistulosum and Colchicum roots with acenaphthene crystals and then wrapped the plants in moist filter paper. After four days of growth, the spindles were altered and the centromeres inactivated: this process has been termed "c-mitosis" because a similar effect occurs with colchicine treat-

ment. Tetraploid and octaploid cells were formed within 14 to 20 days, resulting in the formation of root-tip swellings (c-tumors) in Allium. Mesquita (1967) also investigated the effects of acenaphthene on Allium root cells. He exposed A. cepa root tips to acenaphthene vapor at room temperature for 12 to 96 hours. The reassembling of the phragmoplast elements (small pieces of the endoplasmic reticulum and Golgi bodies) in the equatorial region was inhibited, but the fusion of these elements in other parts of the cell was unimpaired. The result was the random development of cell walls.

To investigate the effect of acenaphthene on mitosis, Dyer (1966) exposed plant species with binucleate pollen (such as Bellevalia romana, Tulbaghia natalensis, and Antirrhinum majus) to vapor from acenaphthene crystals. He found that all cells remained at metaphase, with anaphase being inhibited due to an inhibition of the mitotic spindle. Swanson (1940) also observed effects on mitosis in plant pollen. He scattered acenaphthene crystals on the bottom of a petri dish in which Tradescantia pollen was incubated. The vapors acted by disturbing the spindle mechanism so that the chromosomes remained in place after division. Nebel (1938) examined the effect of acenaphthene on mitosis in plant hairs by treating stamen hairs of Tradescantia with a saturated solution of acenaphthene in liquid media for 2 and 4.5 hours. He found no polyploid cells and no nuclei showing chromosomes in a metaphase condition.

Microorganisms: Several experiments have been performed to investigate the effect of acenaphthene on microorganisms. Hoover

(1972) exposed 37 species of Basidiomycetes to acenaphthene vapors or media containing acenaphthene at unspecified dose levels in order to examine effects on growth, pigment, morphology, nuclear division, and fruit body formation. As the treatment time increased, changes in nuclear division became more pronounced, with a concurrent decrease in the mitotic frequency. Growth, pigment formation, differentiation, and colonial and cellular morphology were affected by acenaphthene treatment. A delay or prevention of light-induced fruitbody formation occurred in one species; two species developed greatly enlarged fruitbodies as a result of this treatment. The genetic stability of these phenotypic changes was not demonstrated, however.

In a later experiment, Hoover and Liberta (1974) exposed hyphae cultures of the fungus Basidiobolus ranarum to acenaphthene vapor for 6 to 18 hours. At the end of 18 hours, gross alterations in nuclear division were observed and the spindle fibers were rendered unstainable. The time required for division was significantly increased in acenaphthene-treated cells. The effect of acenaphthene on fungi was also investigated by Seshadri and Payak (1970). They exposed hyphae of Pythium aphanidermatum to acenaphthene vapor or to a supersaturated solution of acenaphthene for 12 hours. The vapor proved instrumental in arresting the progress of nuclear division. A marked increase in the size and number of sporangia nuclei was noted, and the nuclei showed various degrees of pyknosis and shape irregularity.

Levan and Sandwall (1943) examined the effect of varying concentrations of acenaphthene ($1 \times 10^{-1}$ to $3 \times 10^{-7}$ M solution in

ethanol) on wort yeast cell cultures. Even at the highest concentration, there was no lethality or effect on cell propagation. The authors concluded that the c-mitotic action demonstrated by acenaphthene in higher plants was not observable in yeast. Polyploidy was induced, however, in the yeast Candida scottii (a yeast without a sexual cycle) when treated with 0.2 percent and 1.0 percent acenaphthene added to agar medium (Imshenetsky, et al. 1966). The size of the cell and the nucleus were both increased in the treated cultures, and there was also a higher dry biomass for these cells. The DNA content (µg per cell) was higher in acenaphthene-treated cells, although the difference between experimental and control cultures decreased as the cultures aged.

Imshenetsky and Zhil'tsova (1973) attempted to produce "polyploid-like" cells by exposing Mycobacterium rubrum to vapor from 10 to 20 mg acenaphthene. When the vapor was used alone for treatment, there was no increase in the size of the cells, nor any indication of the induction of polyploidy. When the cells were treated with water or ethylenediaminetetraacetic acid (EDTA) to increase membrane permeability, acenaphthene vapor treatment caused elongation and thickening of cells, with a longer development cycle; these "polyploid-like" changes were found to be unstable, however. In another experiment with bacteria, Avvakumova, et al. (1975) treated Rhizobium (nodule-forming bacteria) with acenaphthene vapor (dose unspecified) to induce polyploidy. The authors found an acenaphthene-associated increase in cellular DNA content and biomass, as well as a change in biochemical properties, e.g., the ability to assimilate carbohydrates and/or organic acids.

Acenaphthene has also been shown to affect mitosis in two species of algae. Sarma and Tripathi (1976a,b) treated Chara globularis and Nitella flagelliformis with a saturated solution of acenaphthene for 12 to 120 hours. The number of cells in mitosis was reduced by 40 percent, and the chromosomes were seen to clump at metaphase after 120 hours. Nine percent of the C. globularis cells showed complete chromosome doubling by the end of the treatment period.

Carcinogenicity

Very little work has been done to determine whether acenaphthene may have carcinogenic properties. Neukomm (1974) reported negative results in a predictive test for carcinogenicity based upon neoplastic induction in the newt Triturus cristatus. Ten animals were injected subcutaneously with acenaphthene (dose and solvent not reported) in the fleshy part of the tail along the vertebral axis. Samples of the injection site were removed at 7 and 14 days, and the tissues were examined for neoplastic infiltration in the epidermis and the development or regression of diffuse tumors. Neoplastic lesions were divided into three categories depending on the size of the lesion and assigned a numerical coefficient accordingly: large (1.0), intermediate (0.5), and limited (0.25). Calculation of a neoplastic index by summing the coefficients of all lesions and dividing by the number of observed animals gave an index for acenaphthene of 0.0, indicating a lack of neoplastic induction in the newt.

Neukomm (1974) discussed the reliability of this test by drawing a correlation between positive index values for a few polycy-

clic aromatic hydrocarbons and the carcinogenicity of these same compounds for mouse skin. These limited comparisons, however, are not sufficient to establish the value of this test for predicting carcinogenicity in mammalian systems.

The only other carcinogenicity studies in the literature involving acenaphthene considered it as one component of a complex mixture of PAH. It is impossible in these studies to sort out the relative contribution of acenaphthene versus other hydrocarbons in the mixture, so no real conclusions can be drawn. Akin, et al. (1976) isolated some polycyclic hydrocarbon-rich fractions of the neutral portion of cigarette smoke condensate (CSC) and tested them for tumor promotion on female mouse skin, using 7,12-dimethylbenz-(a)anthracene (DMBA) as the initiator. Animals were painted once with 125 µg DMBA on dorsal skin; 3 to 4 weeks later the fractions were applied five times a week for 13 months. The fraction containing acenaphthene, pyrene, phenanthrene, and other PAH, showed no significant tumor-promoting activity over controls treated with DMBA and acetone. This result was surprising in view of the fact that Scribner (1973) had demonstrated the tumor-promoting ability of pyrene and phenanthrene.

In 1962, Hoffman and Wynder found that benzene extracts of gasoline exhaust condensates were carcinogenic in mouse skin painting tests. This study is of interest considering a later study by Grimmer, et al. (1977) which showed that acenaphthene was present in an unspecified concentration in the benzene extracts of gasoline

exhaust condensate. Unfortunately, the possible contribution of acenaphthene to the observed carcinogenicity (Hoffman and Wynder, 1962) cannot be determined from this limited evidence.

## CRITERION FORMULATION

### Existing Guidelines and Standards

No existing guidelines or standards were found.

### Current Levels of Exposure

Virtually no information is available concerning the prevalence or concentration of acenaphthene in the environment. Acenaphthene has been detected in cigarette smoke (Harke, et al. 1976; Severson, et al. 1976), automobile exhaust (Grimmer, et al. 1977), and in urban air (Cleary, 1962) and is present in coal tar and several fossil fuel oils. It has also been reported in wastewater from petrochemical, pesticide, and wood preservative industries (U.S. EPA, 1978b) and detected in water from a river in the Netherlands (Meijers and Van der Leer, 1976).

### Special Groups at Risk

Individuals working with coal tar and/or its products face a possible risk due to increased exposure to acenaphthene, although no data are available to estimate this risk.

### Basis and Derivation of Criterion

So little research has been performed on acenaphthene that its mammalian and human health effects are virtually unknown. The two toxicity studies available (Knobloch, et al. 1969; Reshetyuk, et al. 1970) are inadequate for use as the basis of a criterion due to deficiencies in the experimental designs (lack of controls, small number of animals, short durations, etc.). Therefore, until more toxicological data are generated, particularly on genotoxic effects, a criterion based upon organoleptic data is proposed. The lowest levels eliciting human responses were reported to be 0.022

to 0.22 ppm (Lillard and Powers, 1975). Thus, the lower limit 0.02 ppm (0.02 mg/l) appears to be the best estimate of a criterion level that will prevent unpleasant odor from acenapthene. It is emphasized that this criterion is based on aesthetic considerations only and as such has no demonstrated relationship to potential adverse human health effects.

Since the recommended criterion is based on organoleptic effects and is not a toxicological assessment, the consumption of fish and shellfish products was not considered as a route of exposure.

This criterion will be reviewed when additional toxicological data are available.

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